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(FILE 'HOME' ENTERED AT 17:06:33 ON 29 JAN 2003)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 17:07:05 ON 29 JAN 2003

L1 24008 S ELASTIN OR TROPOELASTIN
L2 206836 S VASCULAR(W)STENOSIS OR STENOSIS OR RESTENOSIS OR SVAS OR OBST
L3 536 S L1 AND L2
L4 20040 S (TREAT? OR MODULAT? OR INHIBIT? OR PROPHYLAXIS)(9A)L2
L5 18 S L1(S)L4
L6 10 DUP REM L5 (8 DUPLICATES REMOVED)

=> d bib ab 1-10 l6

L6 ANSWER 1 OF 10 MEDLINE DUPLICATE 1
AN 2001372165 MEDLINE
DN 21321811 PubMed ID: 11428168
TI Extracellular matrix remodeling in the vascular wall.
AU Jacob M P; Badier-Commander C; Fontaine V; Benazzoug Y; Feldman L; Michel J B
CS INSERM U 460, UFR de medecine Xavier Bichat, 16, rue Henri Huchard 75870 Paris, France.. jacob@bichat.inserm.fr
SO PATHOLOGIE BIOLOGIE, (2001 May) 49 (4) 326-32. Ref: 57
Journal code: 0265365. ISSN: 0369-8114.
CY France
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200107
ED Entered STN: 20010730
Last Updated on STN: 20010730
Entered Medline: 20010726
AB The extracellular matrix provides a structural framework essential for the functional properties of vessel walls. The three dimensional organization of the extracellular matrix molecules--**elastin**, collagens, proteoglycans and structural glycoproteins--synthesized during fetal development--is optimal for these functions. Early in life, the vessel wall is subjected to injury: lipid deposition, hypoxia, enzyme secretion and reactive oxygen species production during inflammatory processes, and the extracellular matrix molecules are hydrolyzed by proteases--matrix metalloproteinases, leukocyte elastase, etc. In uninjured arteries and veins, some proteases are constitutively expressed, but through the control of their activation and/or their inhibition by inhibitors, these proteases have a very low activity. During the occurrence of vascular pathologies--atherosclerosis, hypertension, varicosis, **restenosis**, etc.--the balance between proteases and their **inhibitors** is temporally destroyed through the induction of matrix metalloproteinase gene expression or the secretion of enzymes by inflammatory cells. Smooth muscle cells, the most numerous cells in vascular walls, have a high ability to respond to injury through their ability to synthesize extracellular matrix molecules and protease inhibitors. However, the three dimensional organization of the newly synthesized extracellular matrix is never functionally optimal. In some other pathologies--aneurysm--the injury overcomes the responsive capacity of smooth muscle cells and the quantity of extracellular matrix decreases. In conclusion, care should be taken to maintain the vascular extracellular matrix reserve and any therapeutic manipulation of the protease/inhibitor balance must be perfectly controlled, because an accumulation of abnormal extracellular matrix may have unforeseen adverse effects.

L6 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2003 ACS
AN 2000:608602 CAPLUS
DN 133:202978
TI Elastin-based compositions for screening of drugs for treatment of vascular diseases
IN Keating, Mark T.; Li, Dean Y.
PA University of Utah Research Foundation, USA
SO PCT Int. Appl., 79 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000050068	A2	20000831	WO 2000-US2526	20000228
WO 2000050068	A3	20011115		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1175225	A2	20020130	EP 2000-913319	20000228
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
PRAI US 1999-258217	A	19990226		
WO 2000-US2526	W	20000228		

AB The present invention provides screening methods that use organisms or cells that lack function in one or both elastin genes. These methods are useful in identifying drugs for the prevention and treatment of obstructive vascular diseases, such as atherosclerosis, vascular restenosis and transplant arteriopathy. Further, the invention provides pharmaceutical compns. contg. elastin-based compns. that are particularly potent regulators of proliferation, differentiation, and migration of smooth muscle cells in vitro and in vivo. These pharmaceutical compns. and related methods are useful in the prevention and treatment of disorders characterized by diminished capacity to regulate smooth muscle cell function.

L6 ANSWER 3 OF 10 MEDLINE DUPLICATE 2
AN 2001015833 MEDLINE
DN 20459192 PubMed ID: 11003759
TI Surgery for bilateral outflow tract obstruction in elastin arteriopathy.
AU Stamm C; Friehs I; Moran A M; Zurakowski D; Bacha E; Mayer J E; Jonas R A; Del Nido P J
CS Department of Cardiac Surgery, Children's Hospital Boston, Harvard Medical School, Boston, Mass., USA.
SO JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY, (2000 Oct) 120 (4) 755-63.
Journal code: 0376343. ISSN: 0022-5223.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200011
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001101
AB OBJECTIVE: A number of patients with Williams syndrome or other forms of **elastin** arteriopathy have stenoses of pulmonary arteries in addition to supravalvular aortic stenosis. We sought to investigate the effect of the degree of pulmonary arterial stenosis on the prognosis after

an operation for supravalvular aortic **stenosis** to help define the optimal **treatment** strategy for patients with severe forms of **elastin** arteriopathy. METHODS: Between 1960 and 1999, 33 patients underwent operations for supravalvular aortic stenosis while having significant stenoses of the pulmonary arteries. We retrospectively reviewed patient charts, obtained current follow-up information, and determined risk factors for survival and reoperation. RESULTS: Fifteen patients with moderate right-sided obstructions (confirmed by pulmonary artery Z-scores and right ventricular/descending aortic pressure ratio) underwent operations for supravalvular aortic stenosis only. Eighteen patients had more severe right-sided obstructions and underwent surgical relief of pulmonary arterial stenoses or right ventricular outflow tract obstruction in addition to operations for supravalvular aortic stenosis. Eight patients had undergone preoperative balloon dilations of stenotic pulmonary arteries. There were 6 early deaths and 1 late death in our series. Survival at 10 and 20 years was 76% (70% confidence interval, 68%-84%) and freedom from reintervention was 59% (70% confidence interval, 46%-71%) at 10 years and 49% (70% confidence interval, 35%-62%) at 20 years. Multivariate analysis revealed that patients with a right ventricular/descending aortic pressure ratio of 1.0 or more were at higher risk for reintervention but not for death. CONCLUSIONS: Surgical treatment of pulmonary artery obstructions in **elastin** arteriopathy is palliative but, in conjunction with balloon dilation of peripheral pulmonary arteries, offers good long-term survival to patients with the severest form of **elastin** arteriopathy.

L6 ANSWER 4 OF 10 MEDLINE DUPLICATE 3
AN 2000134513 MEDLINE
DN 20134513 PubMed ID: 10666406
TI The insulin-like growth factor axis: A review of atherosclerosis and restenosis.
AU Bayes-Genis A; Conover C A; Schwartz R S
CS Division of Cardiovascular Diseases, Mayo Clinic and Foundation, Rochester, MN 55905, USA.
SO CIRCULATION RESEARCH, (2000 Feb 4) 86 (2) 125-30. Ref: 83
Journal code: 0047103. ISSN: 1524-4571.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200002
ED Entered STN: 20000309
Last Updated on STN: 20010521
Entered Medline: 20000224
AB Insulin-like growth factors I and II (IGF-I and -II) and their regulatory proteins are secreted by cells of the cardiovascular system. They are growth promoters for arterial cells and mediators of cardiovascular disease. IGFs are bound to IGF binding proteins (IGFBPs), which modulate IGF ligand-receptor interaction and consequently to IGF action. IGFBPs are in turn posttranslationally modulated by specific proteases. This dynamic balance (IGFs, IGFBPs, and IGFBP proteases) constitutes the IGF axis and ultimately determines the extent of IGF-dependent cellular effects. Dysregulated actions of this axis influence coronary atherosclerosis through effects on vascular smooth muscle cell growth, migration, and extracellular matrix synthesis in the atherosclerotic plaque. IGF-I promotes macrophage chemotaxis, excess LDL cholesterol uptake, and release of proinflammatory cytokines. Endothelial cells also receive the effects of IGFs stimulating their migration and organization forming capillary networks. Neointimal hyperplasia of **restenosis** after coronary artery injury is also **modulated** by the IGF axis. IGFs stimulate vascular smooth muscle cell proliferation and migration to form the neointima and upregulate **tropoelastin** synthesis after disruption

of the elastic layer. Understanding IGF axis regulation establishes a scientific basis for strategies directed to limit or reverse plaque growth and vulnerability in atherosclerosis and in the neointimal hyperplasia of restenosis.

L6 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2003 ACS
AN 1999:348932 CAPLUS
DN 131:125201
TI Inhibitory mechanisms by which suramin may attenuate neointimal formation after balloon angioplasty
AU Gray, Timothy J.; Strauss, Bradley H.; Hinek, Aleksander
CS Division of Cardiovascular Research, The Hospital for Sick Children, Toronto, ON, M5G 1X8, Can.
SO Journal of Cardiovascular Pharmacology (1999), 33(6), 960-971
CODEN: JCPCDT; ISSN: 0160-2446
PB Lippincott Williams & Wilkins
DT Journal
LA English
AB Restenotic neointimal lesions, a major limitation to coronary angioplasty, develop in response to diverse signals and depend on three properties of activated arterial smooth muscle cells (SMCs): proliferation, migration, and abnormal prodn. of extracellular matrix. Most of the pharmacol. approaches targeting specific pathogenic factors facilitating development of restenosis have failed in clin. trials. Our results indicate that the polysulfonated naphthylurea suramin, a "non-specific drug" that interferes with multiple cellular proteins, inhibits neointimal formation in rabbit iliac arteries after balloon-catheter injury administered throughout the crit. period of several weeks after the procedure. In vitro studies aimed at dissecting the mechanism(s) underlying the suramin-dependent effect demonstrated that, in addn. to an inhibitory effect on SMC proliferation, suramin inhibited fibronectin and elastin deposition and the migration of SMCs through elastin membranes and into scratch gaps of monolayer cultures. We also demonstrated that suramin causes cell-surface accumulation of the elastin binding protein, a receptor that not only anchors SMCs to the extracellular matrix, but also inhibits SMC response to interleukin-1.beta. (IL-1.beta.). We conclude that suramin acts as a multitarget inhibitor of SMC activation and has a therapeutic potential as an agent that may attenuate arterial restenosis after angioplasty.
RE.CNT 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 6 OF 10 MEDLINE DUPLICATE 4
AN 1999344218 MEDLINE
DN 99344218 PubMed ID: 10415729
TI Effect of matrix metalloproteinase inhibition on progression of atherosclerosis and aneurysm in LDL receptor-deficient mice overexpressing MMP-3, MMP-12, and MMP-13 and on restenosis in rats after balloon injury.
AU Prescott M F; Sawyer W K; Von Linden-Reed J; Jeune M; Chou M; Caplan S L; Jeng A Y
CS Metabolic and Cardiovascular Research Department, Novartis Institute for Biomedical Research, Summit, New Jersey 07901, USA..
margaret.prescott@pharma.novartis.com
SO ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1999 Jun 30) 878 179-90.
Journal code: 7506858. ISSN: 0077-8923.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199908
ED Entered STN: 19990820
Last Updated on STN: 20000303
Entered Medline: 19990811
AB The broad-spectrum MMP inhibitor CGS 27023A was tested to determine its potential as a therapy for atherosclerosis, aneurysm, and restenosis. LDL

receptor-deficient (LDLr -/-) mice fed a high-fat, cholic acid-enriched diet for 16 weeks developed advanced aortic atherosclerosis with destruction of elastic lamina and ectasia in the media underlying complex plaques. Lesion formation correlated with a 4.6- to 21.7-fold increase in MMP-3, -12, and -13 expression. Treatment with CGS 27023A (p.o., b.i.d. at 50 mg/kg) had no effect on the extent of aortic atherosclerosis (36 +/- 4% versus 30 +/- 2% in controls), but both aortic medial **elastin** destruction and ectasia grade were significantly reduced (38% and 36%, respectively, $p < 0.05$). In the rat ballooned-carotid-artery model, CGS 27023A (12.5 mg/kg/day via osmotic minipump) reduced smooth muscle cell migration at 4 days by 83% ($p < 0.001$). Intimal lesions were reduced by 85% at 7 days ($p < 0.001$), but intimal smooth muscle proliferation was unaffected, and inhibitory efficacy was lost with time. At 12 days, intimal lesion reduction was less potent (52%, $p < 0.01$). At 3 and 6 weeks, reductions of 11% and 4%, respectively, were not significant. This demonstrates that it is essential to include late time points when the ballooned-carotid-artery model is employed to ensure that lesion size does not "catch up" when a compound solely inhibits smooth muscle cell migration. In summary, MMP inhibitor therapy delayed but did not prevent intimal lesions, thereby demonstrating little promise to prevent **restenosis**. In contrast, MMP **inhibitor** therapy may prove useful to retard progression of aneurysm.

L6 ANSWER 7 OF 10 MEDLINE DUPLICATE 5
AN 1998134362 MEDLINE
DN 98134362 PubMed ID: 9474087
TI Wound healing: a paradigm for lumen narrowing after arterial reconstruction.
AU Geary R L; Nikkari S T; Wagner W D; Williams J K; Adams M R; Dean R H
CS Department of Surgery, Wake Forest University, Winston-Salem, USA.
NC HL25161 (NHLBI)
SO JOURNAL OF VASCULAR SURGERY, (1998 Jan) 27 (1) 96-106; discussion 106-8. Journal code: 8407742. ISSN: 0741-5214.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199803
ED Entered STN: 19980312
Last Updated on STN: 19980312
Entered Medline: 19980305
AB PURPOSE: The intimal hyperplasia hypothesis that equates lumen narrowing after arterial injury with intimal mass has recently been challenged. Evidence has emerged to suggest that lumen narrowing is caused in large part by changes in artery wall geometry rather than intimal mass per se. We have begun to explore this hypothesis in a unique nonhuman primate model of atherosclerosis. METHODS: Monkeys who were fed an atherogenic diet for 3 to 5 years underwent experimental angioplasty of the left iliac artery. The contralateral iliac artery served as an intraanimal control. Arteries were removed 2, 4, 7, 14, 28, or 112 days later for analysis (6 or 13 per time point). Angioplasty dilated arteries by fracturing atheroma and stretching or tearing the media. Cross-sections of injured arteries were analyzed for expression of extracellular matrix components and cell surface integrins that are important in wound healing. Antibodies, riboprobes, or histochemical stains specific for fibrin, hyaluronan, versican (chondroitin sulfate-containing proteoglycan), procollagen-I, **elastin**, and the alpha 2 beta 1 and alpha V beta 3 integrins were used. RESULTS: A thin mural thrombus was seen at sites of denudation and plaque fracture (days 2 to 7). This provisional matrix was invaded by leukocytes (days 2 to 4) and alpha-actin-positive smooth muscle cells (SMCs; days 4 to 7). Thrombus was replaced by SMCs expressing hyaluronan and the associated versican proteoglycans (day 14). Versican was expressed throughout the neointima as it enlarged (day 28), but expression later subsided (day 112). Procollagen-I expression initially increased in the

adventitia (day 4) and then in the forming neointima (day 14). Procollagen-I expression was found to persist within the adventitia and in the neointima in SMCs nearest the lumen (days 28 to 112). **Elastin** staining was prominent within the mature neointima (day 112) but not at earlier time points. Integrin expression also increased within the injured artery wall. alpha v beta 3 staining (fibrin[ogen] receptor) increased in the injured media (days 2 to 7) and was then seen throughout the early neointima (day 7). Low level expression of alpha V beta 3 subsequently persisted within the forming neointima (day 28). alpha 2 beta 1 (collagen receptor) expression increased in the neointima in SMCs nearest the lumen (day 28). CONCLUSIONS: Lumen narrowing after angioplasty in this model of atherosclerosis is caused largely by decreased artery wall diameter. The pattern of matrix and integrin expression within the injured artery wall is in many ways analogous to that of healing wounds. These observations suggest that tissue contraction may play a role in lumen narrowing at sites of arterial reconstruction. Strategies to **inhibit** wound contraction may prove effective in preventing **restenosis**.

L6 ANSWER 8 OF 10 MEDLINE DUPLICATE 6
AN 1998144430 MEDLINE
DN 98144430 PubMed ID: 9483436
TI [Ultrasound coronary angioplasty: state of the art and new clinical aspects].
Koronare Ultraschallangioplastie: Standpunkt und neue klinische Aspekte.
AU Rosenschein U; Budde-Schwartzman B
CS Department of Cardiology, Tel Aviv Sourasky Medical Center, Israel.. angioyt@netvision.net.il
SO HERZ, (1997 Dec) 22 (6) 308-17. Ref: 24
Journal code: 7801231. ISSN: 0340-9937.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA German
FS Priority Journals
EM 199805
ED Entered STN: 19980514
Last Updated on STN: 19980514
Entered Medline: 19980505
AB Therapeutic ultrasound was shown to ablate thrombi and to disrupt atherosclerotic plaques in vitro and recently to recanalize occluded coronary arteries in acute myocardial infarction (AMI). The goal of this article is to update collective experience and to weigh the promising and unresolved aspects of this newly developed technology and its clinical results. As therapeutic ultrasound was for long known a synonym for lithotripsy of calculi diseases, it lastly received high attention as a catheter-based ultrasound method to ablate thrombi and disrupt atherosclerotic plaques in interventional cardiology (Figure 1). The effect of therapeutic ultrasound to ablate selectively pathological tissue depends on its bioselectivity for elastic fibers: After ultrasound sonication, healthy tissue-rich in **elastin** and collagen-including arterial wall remains intact whereas thrombus and plaque with their minimal elastic support are found to be highly susceptible to ablation. Our catheter for coronary ultrasound thrombolysis (Figure 2) consists of a solid metal probe and is connected to a piezo-electric transducer at its proximal end. The distal part ends in a three-wire flexible segment with a 1.6 mm tip ball to guarantee maximal wire flexibility and optimal transmission of ultrasound energy. The initial in vitro studies resulted in a fundamental understanding of the destructive effect of ultrasound on tissue based on 4 factors: mechanical vibration, thermal effects, microcurrents, and cavitation. The first studies on human peripheral vessels were published in 1991 being performed during femoral bypass surgery on occluded and partially obstructed arteries. The procedure was performed without perforation, no adverse side

effects emerged, restenosis rate was 20%. The clinical application of coronary ultrasound angioplasty was initiated in 1991; Siegel published his data on 44 patients. In his study, 30 patients with chronic atherosclerotic occlusive lesions and 14 with unstable or stable angina or AMI were **treated** by ultrasound angioplasty. Residual **stenosis** after ultrasound **treatment** was 71%, after balloon dilation reduced to 34%. In the 6-month follow-up angiograms showed no major adverse effect or restenosis. Our experience with coronary ultrasound thrombolysis (CUT) is based on the analysis of 33 patients' data in the feasibility (Table 1) plus multicenter phase of the ACUTE trial (Analysis of Coronary Ultrasound Thrombolysis Endpoints) (Figure 3). Our patients were exclusively treated for AMI by ultrasound angioplasty and afterwards by PTCA if required (Figure 4). The average final percent stenosis was 20% (Figure 5). The main efficacy parameters, device success and angiographic success rates were 100%, clinical success rate was 91.7% (Figure 6 and Table 2). The adverse clinical events of CUT are limited--at least in our studies--to reocclusion of infarct-related artery and ischemia and could be reversed by additional PTCA. No adverse clinical side effects were observed during sonication of the coronary tree. Final angiography revealed residual stenosis of 20% without morphological signs. These excellent results suggest that bioselectivity of ultrasound together with the developed skills of the catheter system induces rapid and selective thrombolysis with no need to cross the target lesion before sonication. But what is the better solution for thrombosis and which for plaque disruption? The development of transluminal balloon catheter really modified therapeutic approach to obstructive coronary and peripheral arterial disease but it is still accompanied by a high rate of abrupt closure, AMI and death. Although the use of intravenous thrombolytic agents is well established in the treatment of AMI and these agents are widely used, a large patient collective remains (up to 33% and more) in whom their use is inadvisable due to recent stroke, surgery, trauma or other contraindications. (ABSTRACT TRUNCATED)

L6 ANSWER 9 OF 10 SCISEARCH COPYRIGHT 2003 ISI (R)
AN 94:515010 SCISEARCH
GA The Genuine Article (R) Number: PC076
TI SUPRAVALVAR AORTIC-STENOSIS
AU FRIEDMAN W F (Reprint)
CS UNIV CALIF LOS ANGELES, SCH MED, DEPT PHYS, 22-412 MDCC, LOS ANGELES, CA, 90024 (Reprint)
CYA USA
SO PROGRESS IN PEDIATRIC CARDIOLOGY, (AUG 1994) Vol. 3, No. 3, pp. 133-139. ISSN: 1058-9813.
DT Article; Journal
FS CLIN
LA ENGLISH
REC No References Keyed
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB New information suggests that a genetic defect for supravalvar aortic stenosis may be located in the same chromosomal subunit as **elastin** on chromosome 7. The two most common clinical presentations are patients with an autosomal dominant familial pedigree associated with normal facies and intelligence, and others with the nonfamilial Williams syndrome with abnormal facial appearance and mental retardation. Peripheral pulmonary arterial stenosis often coexists with the supravalvar aortic obstruction in both of these patient groups. Transcatheter **treatment** of supravalvar aortic **stenosis** is not likely to be effective. With severe obstruction, surgical patch procedures are indicated if the aorta is not markedly hypoplastic. Newer operative patch procedures may improve long-term results.

L6 ANSWER 10 OF 10 MEDLINE
AN 92025826 MEDLINE
DN 92025826 PubMed ID: 1927864

TI 476 nm excited laser-induced fluorescence spectroscopy of human coronary arteries: applications in cardiology.
AU Richards-Kortum R; Rava R P; Fitzmaurice M; Kramer J R; Feld M S
CS G. R. Harrison Spectroscopy Laboratory, Massachusetts Institute of Technology, Cambridge 02139.
NC RR02594 (NCRR)
SO AMERICAN HEART JOURNAL, (1991 Oct) 122 (4 Pt 1) 1141-50.
Journal code: 0370465. ISSN: 0002-8703.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199110
ED Entered STN: 19920124
Last Updated on STN: 19920124
Entered Medline: 19911031
AB We have shown that normal coronary arteries and noncalcified and calcified atherosclerotic plaque can be differentiated on the basis of the 476 nm excited fluorescence spectra, providing the basis of a spectroscopic guidance system for coronary artery laser angiosurgery. This discrimination is based on extraction of parameters from tissue fluorescence spectra, which are proportional to the tissue concentrations of structural proteins (collagen and **elastin**) and ceroid via a model of tissue fluorescence. We use these parameters to calculate the likelihood that an area of interest in a coronary artery is normal, noncalcified, or calcified plaque. This method of diagnosing atherosclerosis provides information about the histochemical composition of atherosclerotic lesions and is thus fundamentally different from the diagnostic methods currently used. It may ultimately have bearing on a number of pertinent clinical problems. We have discussed applications to studying initiating factors in formation and progression of plaque, healing after interventional **treatments**, and the likelihood of **restenosis** after PTCA.

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